

Leukocyte Chemotactic Factor 2 (LECT2) Amyloidosis in First Nations People in British Columbia, Canada: A Case Series

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BACKGROUND: Leukocyte chemotactic factor 2 (LECT2) amyloidosis was first identified in 2008, and has emerged as a frequent type of renal amyloidosis. It is typically reported as being renal limited, and, in the United States, more prevalent in Hispanic patients. We report 4 First Nations people living in Northern British Columbia who were diagnosed with renal LECT2 amyloidosis over the past 4 years.

METHODS: All patients presented with slowly progressive renal impairment and minimal proteinuria. (Table 1)

Age	Sex	Creatinine $\mu\text{mol/L}$	eGFR (ml/min)	ACR (mg/mmol)
56	F	167	37	1.9
78	M	261	21	64
68	F	223	20	48
62	F	165	27	2.4

Biopsy findings were typical of LECT2 amyloid, with intense congo red staining, and amyloid deposition in the renal interstitium and vasculature as well as glomeruli. After immunohistochemistry for common amyloidogenic proteins did not identify the pathogenic protein, laser microdissection-mass spectrometry was used to make the diagnosis. Although First Nations people comprise only about 4% of the patient population seen by our Nephrology service, all 4 cases of renal LECT2 amyloidosis, diagnosed over the past 4 years, occurred in this ethnic group.

CONCLUSIONS: The pathogenesis of LECT2 amyloidosis is currently not well understood. Sequencing of the coding region of the *LECT2* gene in patients with LECT2 amyloidosis has revealed a common homozygous single nucleotide polymorphism, indicating a probable genetic component to disease pathogenesis. The fact that our centre has only identified LECT2 amyloidosis in First Nations people adds weight to the hypothesis that there is a genetic contribution to the disease. It may be that a common North American indigenous ancestry of First Nations people and Hispanics accounts for the occurrence of this condition in both populations. LECT2 amyloidosis may be an underdiagnosed cause of chronic kidney disease, as the characteristic

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minimal proteinuria and slow progression of renal impairment probably result in relatively few patients undergoing renal biopsy.